

Permanence and Stability of a Kill the Winner Model in Marine Ecology

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Abstract

We focus on the long term dynamics of “killing the winner” Lotka-Volterra models of marine communities consisting of bacteria, virus, and zooplankton. Under suitable conditions, it is shown that there is a unique equilibrium with all populations present which is stable, the system is permanent, and the limiting behavior of its solutions is strongly constrained.

keywords: Virus, bacteria, zooplankton, kill the winner, infection network, Lotka-Volterra system, permanence.

1 Introduction

It is now known that the microbial and viral communities in marine environments are remarkably diverse but are supported by relatively few nutrients in very limited concentrations [12, 18]. What can explain the observed diversity? What prevents the most competitive bacterial strains from achieving large densities at the expense of less competitive strains? Thingstad [14, 15, 16, 17] has suggested that virus impose top down control of bacterial densities. Together with various coauthors, he has described an idealized food web consisting of bacteria, virus and zooplankton to illustrate mechanisms of population control, referred to as “killing the winner” since any proliferation of a “winning” bacterial strain results in increased predation by some virus. The kill the winner (KtW) mathematical model of this scenario, in the form of a system of Lotka-Volterra equations for bacterial, virus, and zooplankton densities is, as noted by Weitz [18], based on the

assumptions that (1) all microbes compete for a common resource, (2) all microbes, except for one population, are susceptible to virus infection, (3) all microbes are subjected to zooplankton grazing, (4) viruses infect only a single type of bacteria.

Various forms of the KtW model have appeared in the work of Thingstad et al. [14, 15, 16, 17] and recently in the monograph of Weitz [18]. As the nutrient level can be assumed to be in quasi-steady state with consumer densities, the models typically involve only the n bacteria types, $n - 1$ virus types, and a single zooplankton. While the literature contains many numerical simulations of KtW solutions, very little is known about the long term behavior of these solutions. It is the aim of this paper to initiate a mathematical analysis of this important model system. We will show that the equilibrium with all populations present is unique and stable to small perturbations, that the system is permanent in the sense that all population densities are ultimately bounded away from extinction by an initial condition independent positive quantity, and that the long-term average of each population's density is precisely equal to its corresponding positive equilibrium value. In addition, we are able to provide some qualitative information about the long term dynamics. It is shown that the zooplankton density and the density of the bacterial strain resistant to virus infection converge to their equilibrium value. Furthermore, if a solution does not converge to the positive equilibrium, then its long-term dynamics can be described by an uncoupled system consisting of $n - 1$ conservative two-species systems involving each virus-susceptible bacteria and its associated virus. This implies that non-convergent solutions are, at worst, quasi-periodic.

Thingstad notes in [15] that a weakness of the killing the winner hypothesis is the assumption (4) that each virus infects only a single type of bacteria. Indeed, recent data [1, 2, 18] suggests that some virus have large host range. We will also show that most of our conclusions stated above hold without the restriction (4). For example, they hold for a nested infection network.

The results described above allow one to determine a plausible route by which a KtW community (satisfying (1) – (4)) consisting of n bacterial strains, $n - 1$ virus strains, and a single non-specific zooplankton grazer might be assembled starting with a community consisting of a single bacteria and its associated virus and subsequently adding one new population at a time until the final community is achieved. By a plausible route, we require that each intermediate community be

permanent [5], also called uniformly persistent [11, 13, 10], since a significant time period may be required to make the transition from one community to the next in the succession and therefore each community must be resistant to extinctions of its members. In [6, 7], considering only bacteria and virus communities, we established a plausible route to the assembly of a community consisting of n bacterial strains and either n or $n - 1$ virus strains in which the infection network is one to one under suitable conditions. See also [3] although they did not infer permanence. Therefore, since we merely need to add zooplankton to community consisting of n bacteria and $n - 1$ virus, the main result of this paper ensures that there is a plausible assembly path to the KtW community.

In the next section, we formulate our KtW model and state our main results. Technical details are include in a final section.

2 The KtW model

Our KtW model, consisting of $n \geq 2$ bacterial types, $n - 1$ virus types and one zooplankton, is patterned after equations (7.28) in [18] with slight changes. Densities of bacteria strains are denoted by B_i , virus strains by V_i , and zooplankton by Y . The difference in our model and (7.28) is in the way that inter and intra-specific competition among bacteria is modeled. We assume that the density dependent reduction in growth rate due to competition is identical for all bacterial strains as in [6, 8, 7]. Virus strain V_i infects bacterial strain B_i for $i \neq n$ but B_n is resistant to virus infection. Zooplankton graze on bacteria at a strain independent rate. Virus adsorption rate is ϕ_i and burst size is β_i ; w represents a common loss rate. The equations follow.

$$\begin{aligned} B'_i &= B_i(r_i - w - aB) - B_i\phi_iV_i - \alpha B_iY, \\ V'_i &= V_i(\beta_i\phi_iB_i - k_i - w), \quad 1 \leq i \leq n - 1 \\ B'_n &= B_n(r_n - w - aB) - \alpha B_nY \\ Y' &= Y(\alpha\rho B - w - m), \end{aligned} \tag{1}$$

where $B = \sum_j B_j$ is the sum of all bacterial densities.

It is convenient to scale variables as:

$$P_i = \phi_iV_i, \quad H_i = aB_i, \quad Z = \alpha Y,$$

and parameters as

$$n_i = \beta_i \phi_i / a, \quad \lambda = \alpha \rho / a, \quad e_i = \frac{k_i + w}{n_i}, \quad q = \frac{w + m}{\lambda}.$$

This results in the following scaled system where $H = \sum_j H_j$:

$$\begin{aligned} H'_i &= H_i(r_i - w - H) - H_i P_i - H_i Z, \\ P'_i &= n_i P_i(H_i - e_i), \quad 1 \leq i \leq n-1 \\ H'_n &= H_n(r_n - w - H) - H_n Z \\ Z' &= \lambda Z(H - q). \end{aligned} \tag{2}$$

Only positive solutions of (2) with $H_i(0) > 0, P_j(0) > 0, Z(0) > 0$ for all i, j are of interest. It is then evident that $H_i(t) > 0, P_j(t) > 0, Z(t) > 0$ for all t and i, j .

There is a unique positive equilibrium E^* if and only if the virus-resistant microbe H_n has the lowest growth rate among the bacteria

$$w < r_n < r_j, \quad 1 \leq j \leq n-1, \tag{3}$$

and if

$$\sum_{i=1}^{n-1} e_i < q < r_n - w. \tag{4}$$

Then E^* is given by

$$H_j^* = e_j, P_j^* = r_j - r_n, \quad j \neq n, \quad H_n^* = q - \sum_{i=1}^{n-1} e_i, \quad Z^* = r_n - w - q.$$

Evidently, (4) requires that each virus strain controls the population density of its targeted bacterial strain such that the zooplankton cannot be maintained without the presence of the resistant strain, which cannot grow too slowly.

Our main result follows. We assume that (3) and (4) hold.

Theorem 1. *E^* is a stable equilibrium and the system is permanent in the sense that there exists $\epsilon > 0$ such that every positive solution satisfies:*

$$H_i(t) > \epsilon, \quad P_j(t) > \epsilon, \quad Z(t) > \epsilon, \quad t > T \tag{5}$$

for all i, j where $T > 0$, but not ϵ , depends on initial conditions.

The long term time average of each population is its equilibrium value:

$$\lim_{t \rightarrow \infty} \frac{1}{t} \int_0^t X(s) ds = X^*, \quad X = H_i, P_j, Z, \quad (6)$$

and $H_n(t)$ and $Z(t)$ converge to their equilibrium values H_n^* and Z^* .

Moreover, a positive solution either converges to E^* or its omega limit set consists of non-constant positive entire trajectories satisfying $\sum_{i=1}^n H_i(t) = \sum_{i=1}^n H_i^*$, $H_n(t) = H_n^*$, $Z(t) = Z^*$, and where $(H_i(t), P_i(t))$ is a positive solution of the classical Volterra system

$$\begin{aligned} H_i' &= H_i(P_i^* - P_i) \\ P_i' &= n_i P_i (H_i - H_i^*), \quad 1 \leq i \leq n-1. \end{aligned} \quad (7)$$

As advertised in the introduction, Theorem 1 says that the KtW equilibrium is unique and stable to perturbations. More importantly, the system is permanent in the sense that all population densities are ultimately bounded away from extinction by an initial condition independent positive quantity. The zooplankton density and the density of the bacterial strain resistant to virus infection converge to their equilibrium values and if a solution does not converge to the positive equilibrium, then its long-term dynamics is described by the system consisting of $n-1$ conservative two-species systems (7). The latter would imply that H_i, P_i are periodic with period depending on parameters and its amplitude. However, the restriction $\sum_{i=1}^n H_i(t) = \sum_{i=1}^n H_i^*$ requires a very special resonance among the periods, suggesting that this alternative is unlikely.

Of course, our KtW model (1) is very special. Our aim was not to offer a general KtW model. Rather, it was to say as much as we could about the long term dynamics of a KtW model and for that, we made simplifying assumptions. Most of these assumptions are also made in the system (7.28) in [18] and in similar models in the literature [1, 2]. It should be noted that our main result, that the KtW model is permanent, continues to hold for sufficiently small changes in system parameters [11].

Finally, we note that the main results of our earlier work [8], in which we were concerned only with bacteria-virus infection networks, can be applied to obtain results similar to Theorem 1 for KtW models with more general infection networks than the one to one network. For example, our scaled model for the nested infection network consisting

of n bacteria strains and n virus strains in [8] is the following:

$$\begin{aligned} H'_i &= H_i \left(r_i - \sum_{j=1}^n H_j - \sum_{j \geq i} P_j \right) \\ P'_i &= e_i n_i P_i \left(\sum_{j \leq i} H_j - \frac{1}{e_i} \right), \quad 1 \leq i \leq n. \end{aligned} \quad (8)$$

To compare with (2), set $Z = P_n$ and regard it as a zooplankton grazer. Also, we must view the r_i as $r_i - w$, $n_i = k_i + w$, and $e_i = \beta_i \phi_i / n_i$, viewed as the efficiency of virus infection of bacteria, is comparable to the reciprocal of its value in (2). The existence of a positive equilibrium for (8) requires life history trade-offs of bacteria and virus strains. Bacteria that are more susceptible to virus infection must grow faster

$$r_1 > r_2 > \cdots > r_n > Q_n, \quad (9)$$

and the efficiency of virus infection should decline as its host range increases:

$$e_1 > e_2 > e_3 > \cdots > e_n. \quad (10)$$

Here, $Q_n = \frac{1}{e_1} + \left(\frac{1}{e_2} - \frac{1}{e_1} \right) + \left(\frac{1}{e_3} - \frac{1}{e_2} \right) + \cdots + \left(\frac{1}{e_n} - \frac{1}{e_{n-1}} \right)$. If (9) and (10) hold, there is a unique positive equilibrium E^* and all positive solutions converge to it [8]. By simply renaming $Z = P_n$ and regarding it is a zooplankton, we obtain an even stronger result than Theorem 1 for the KtW model with nested infection network provided these tradeoffs hold. Quite arbitrary infection networks among bacteria and phage might be treated using the approach in [9].

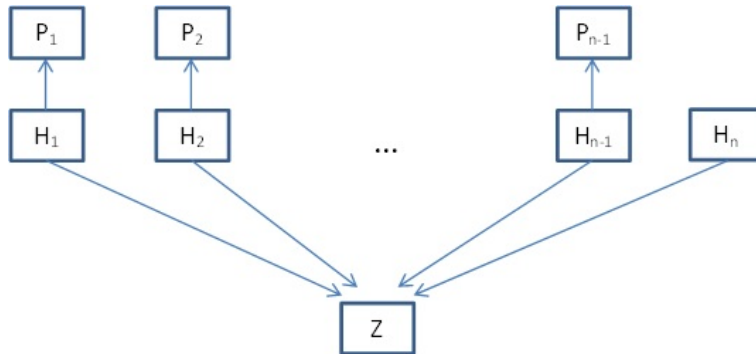


Figure 1: Interactions between the $n-1$ virus, n host, and the zooplankton.

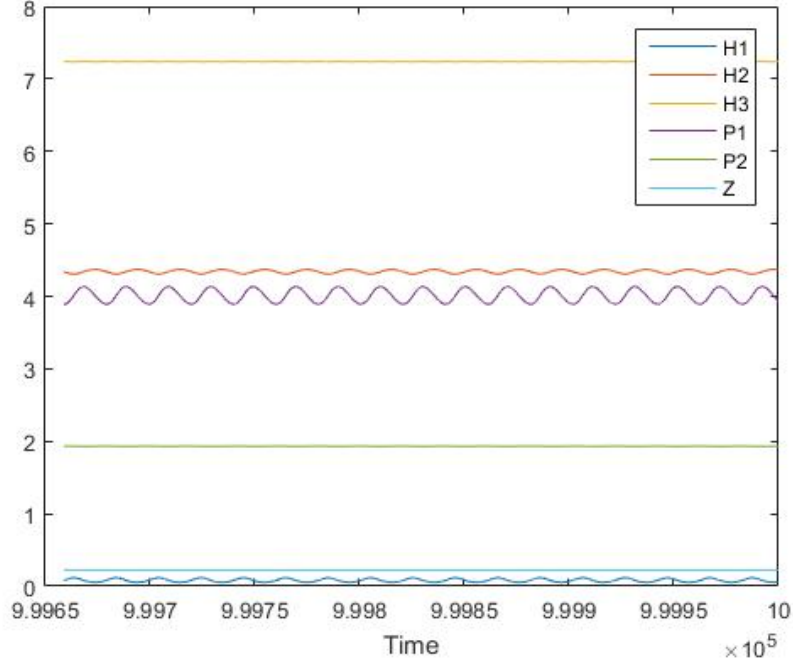


Figure 2: Last 1000 time units of a 1 million run on a population of 3 bacteria, 2 virus, and one zooplankton using ode45. Parameters specified in the figure are chosen to satisfy conditions (3), and (4), and are not intended to be biologically realistic. Solutions are highly oscillatory, and seem to be periodic.

3 Proof of Main Result

Proposition 2. *Solutions of (2) with nonnegative (positive) initial data are well-defined for all $t \geq 0$ and remain nonnegative (positive). In addition, the system has a compact global attractor. Indeed, if*

$$F(t) = \sum_{i=1}^n H_i(t) + \sum_{i=1}^{n-1} \frac{P_i(t)}{n_i} + \frac{Z}{\lambda} \text{ then}$$

$$F(t) \leq \frac{Q}{W} + (F(0) - \frac{Q}{W})e^{-Wt} \leq \max\{F(0), \frac{Q}{W}\},$$

and

$$\limsup_{t \rightarrow \infty} F(t) \leq \sum_{i=1}^n (1 + \frac{r_i}{W}) r_i,$$

where $K = \max_{i=1}^n \{H_i(0), r_i\}$, $W = \min_{i=1}^n \{e_i, w, q\}$ and $Q = \sum_{i=1}^n (W + r_i) K$.

Proof. Existence and positivity of solutions follow from the form of the right hand side. Therefore, $H'_i(t) \leq H_i(t)(r_i - H_i(t))$. Hence $H_i(t) \leq K$ and $\limsup_{t \rightarrow \infty} H_i(t) \leq r_i$.

$$\begin{aligned} \frac{dF}{dt} &= \sum_{i=1}^n (r_i - w) H_i - \left(\sum_{i=1}^n H_i \right) \left(\sum_{j=1}^n H_j \right) - \sum_{i=1}^n P_i e_i - Zq \\ &\leq \sum_{i=1}^n r_i H_i - W \sum_{i=1}^n \left(H_i + \frac{P_i}{e_i n_i} + Z \right) \\ &= \sum_{i=1}^n (W + r_i) H_i - WF. \end{aligned}$$

The estimate on $F(t)$ follows by bounding the first summation by Q and integrating; the estimate on the limit superior follows from the estimate of the limit superior of the H_i above and by integration. \square

Proof. Proof of Theorem 1. Since positive equilibrium E^* exists, we can write (2) as

$$\begin{aligned} H'_i &= H_i \left(\sum_{j=1}^n (H_j^* - H_j) + P_i^* - P_i + Z^* - Z \right) \\ H'_n &= H_n \left(\sum_{j=1}^n (H_j^* - H_j) + (Z^* - Z) \right) \\ P'_i &= n_i P_i (H_i - H_i^*), \quad 1 \leq i < n \\ Z' &= Z \lambda \sum_{i=1}^n (H_i - H_i^*) \end{aligned} \tag{11}$$

Let $U(x, x^*) = x - x^* - x^* \log x/x^*$, $x, x^* > 0$, be the familiar Volterra function and let

$$V(H, P, Z) = \sum_{i=1}^n c_i U(H_i, H_i^*) + \sum_{j=1}^m d_j U(P_j, P_j^*) + g U(Z, Z^*) \tag{12}$$

where c_1, \dots, c_n and d_1, \dots, d_m and g are to be determined.

Then the derivative of V along solutions of (11), \dot{V} , is given by

$$\begin{aligned}\dot{V} = & - \left(\sum_i^n c_i (H_i - H_i^*) \right) \left(\sum_j^n (H_j - H_j^*) \right) \\ & - \sum_i^{n-1} c_i (H_i - H_i^*) (P_i - P_i^*) \\ & - \sum_i^n c_i (H_i - H_i^*) (Z - Z^*) + \sum_i^{n-1} d_i n_i (P_i - P_i^*) (H_i - H_i^*) \\ & + \sum_i^n g \lambda (Z - Z^*) (H_i - H_i^*)\end{aligned}$$

If $c_i = 1$, $g = \frac{1}{\lambda}$, and $d_i = \frac{1}{n_i}$ then the last four summations cancel out and we have

$$\dot{V} = - \left(\sum_i H_i - \sum_i H_i^* \right)^2 \quad (13)$$

As $\dot{V} \leq 0$, E^* is locally stable [4] and for each positive solution there exists $p, P > 0$ such that $p \leq x(t) \leq P, t \geq 0$, where $x = H_i, P_j, Z$. Then (6) follows immediately from Theorem 5.2.3 in [5].

Consider a positive solution of (11). By LaSalle's invariance principle [4, 5], every point in its omega limit set L must satisfy $\sum_i H_i(t) = \sum_i H_i^*$ since $L \subset \{(H, V) : \dot{V} = 0\}$. Since $V(x) \leq V(H(0), P(0))$ for all $x \in L$, L is a compact subset of the interior of the positive orthant. We now consider a trajectory belonging to L ; until further notice, all considerations involve this solution. Since $\sum_i H_i(t) = \sum_i H_i^*$, the solution satisfies

$$\begin{aligned}H_i' &= H_i (P_i^* - P_i + Z^* - Z) \\ P_i' &= n_i P_i (H_i - H_i^*), \quad 1 \leq i \leq n-1 \\ H_n' &= H_n (Z^* - Z) \\ Z' &= 0\end{aligned} \quad (14)$$

We see that $Z' \equiv 0$, therefore $Z(t)$ is a constant. Then, $H_n' = H_n (Z^* - Z)$ so $H_n(t)$ either converges to zero, blows up to infinity, or is identically constant, depending on the value of Z . The only alternative consistent with L being invariant, bounded, and bounded away

from the boundary of the orthant is that $H_n(t)$ is constant and that $Z = Z^*$. By (6), it follows that $H_n = H_n^*$. Therefore (14) becomes:

$$\begin{aligned} H'_i &= H_i(P_i^* - P_i) \\ P'_i &= n_i P_i(H_i - H_i^*), \quad 1 \leq i \leq n-1 \\ H_n &= H_n^* \\ Z &= Z^* \end{aligned} \tag{15}$$

This establishes the assertions regarding (7). Note that as (15) holds on the limit set L of our positive solution, it follows that $H_n(t) \rightarrow H_n^*$, $Z(t) \rightarrow Z^*$ for our positive solution.

Finally, we prove (5). It follows from (6) that $\limsup_{t \rightarrow \infty} x(t) = x^*$, for each component $x = H_i, P_j, Z$ of an arbitrary positive solution of (11). This means that (11) is uniformly weakly persistent. Proposition 2 implies that the key hypotheses of Theorem 4.5 from [13, 10] are satisfied, and therefore weak uniform persistence implies strong uniform persistence. This is precisely (5). \square

Parameter	value
r_1	17.089453152634810
r_2	15.009830525061846
r_3	13.077955412892173
n_1	0.299362132425990
n_2	0.011514418415303
e_1	0.081255501212170
e_2	4.340892914457329
q	10.465564663600418
λ	4.474468552537804
w	1.194565710732100

Figure 3: Parameter values used in *Figure 2*

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